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=> d his

     (FILE 'HOME' ENTERED AT 14:14:09 ON 17 SEP 2004)

     FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, SCISEARCH' ENTERED AT 14:15:38 ON
     17 SEP 2004
                E WOODS GORDON/AU
L1           46 S E3, E5
L2            2 S L1 AND (CADMIUM OR CD)
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=> s l1 and (cadmium or cd)
L2            2 L1 AND (CADMIUM OR CD)

=> d ibib abs kwic 1-2

L2   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:         2002:638144  CAPLUS
DOCUMENT NUMBER:          137:163841
TITLE:                    Methods for regulating levels of zinc, cadmium
                          , and calcium in humans and for diagnosing, or
                          screening for the risk of developing diseases
                          associated with abnormal levels of cadmium,
                          zinc and calcium in body fluids and tissues
INVENTOR(S):              Woods, Gordon L.
PATENT ASSIGNEE(S):       USA
SOURCE:                   U.S. Pat. Appl. Publ., 19 pp., Cont.-in-part of U.S.
                          Ser. No. 610,538, abandoned.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   3
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ----------------     ----   --------    --------------------    --------
     US 2002114848         A1    20020822    US 2001-989674          20011121
     WO 2003045404         A2    20030605    WO 2002-US37248         20021121
     WO 2003045404         A3    20031030
         W:  CA
         RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT,
             LU, MC, NL, PT, SE, SK, TR
PRIORITY APPLN. INFO.:                        US 1999-142926P     P  19990709
                                              US 2000-610538      B2 20000707
                                              US 2001-989674      A  20011121
AB   Methods and compns. are provided for decreasing PGE2:PGF2α,
     regulating ratios of zinc:cadmium and regulating the concentration of
     zinc, calcium and zinc-containing and PGE2-dependent matrix metalloproteinases
     in body fluids and tissues of a human.  Elevated or otherwise unregulated
     levels of PGE2, zinc and calcium and elevated concns. of zinc-containing and
     PGE2-dependent matrix metalloproteinases have been found to be associated
     with the development of certain diseases.  Methods for the prevention of a
     variety of diseases are also disclosed.
TI   Methods for regulating levels of zinc, cadmium, and calcium in
     humans and for diagnosing, or screening for the risk of developing
     diseases associated with abnormal levels of cadmium, zinc and
     calcium in body fluids and tissues
IN   Woods, Gordon L.
AB   Methods and compns. are provided for decreasing PGE2:PGF2α,
     regulating ratios of zinc:cadmium and regulating the concentration of
     zinc, calcium and zinc-containing and PGE2-dependent matrix metalloproteinases
     in body fluids and tissues of a human.  Elevated or otherwise unregulated
     levels of PGE2, zinc and calcium and elevated concns. of zinc-containing and
     PGE2-dependent matrix metalloproteinases have been found to be associated
     with the development of certain diseases.  Methods for the prevention of a
     variety of diseases are also disclosed.
ST   zinc cadmium calcium regulation diagnosis therapy; PGE2
     PGF2alpha ratio regulation matrix metalloproteinase body fluid
IT   Intestine, neoplasm
        (colon; zinc, cadmium, and calcium level regulation in
        humans, and use in disease diagnosis and prevention)
IT   Estrogens
     RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
     (Biological study); USES (Uses)
        (conjugated; zinc, cadmium, and calcium level regulation in
        humans, and use in disease diagnosis and prevention)
IT   Drug delivery systems
        (inhalants; zinc, cadmium, and calcium level regulation in
        humans, and use in disease diagnosis and prevention)
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IT Sperm
(motility; zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT Drug delivery systems
(oral; zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT Drug delivery systems
(parenterals; zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT Semen
(plasma; zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT Cell migration
(sperm motility; zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT Osteoporosis
(therapeutic agents; zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT Alzheimer's disease
Animal tissue
Anti-Alzheimer's agents
Antidiabetic agents
Antihypertensives
Antitumor agents
Blood analysis
Blood serum
Body fluid
Diabetes mellitus
Diagnosis
Drug bioavailability
Drug delivery systems
Equus caballus
Erythrocyte
Human
Hypertension
Mammary gland, neoplasm
Osteoporosis
Prostate gland, neoplasm
Semen
Urine
Urine analysis
(zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT Calcium channel
Prostate-specific antigen
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT Estrogens
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT 363-24-6, PGE2 551-11-1, PGF2α 7440-43-9, **Cadmium**, biological studies 7440-66-6, Zinc, biological studies 7440-70-2, Calcium, biological studies 141907-41-7, Matrix metalloproteinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)
IT 543-90-8, **Cadmium** acetate 7440-43-9D, **Cadmium**, salts 10108-64-2, **Cadmium** chloride 10124-36-4, **Cadmium** sulfate 10325-94-7, **Cadmium** nitrate 149845-06-7, Invirase 155213-67-5, Ritonavir 157810-81-6, Indinavir sulfate 159989-65-8, Nelfinavir mesylate
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(zinc, **cadmium**, and calcium level regulation in humans, and use in disease diagnosis and prevention)

L2 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:50495 CAPLUS
DOCUMENT NUMBER: 134:95488
TITLE: **Cadmium** containing compositions for prevention and treatment of prostate cancer
INVENTOR(S): **Woods, Gordon L.**
PATENT ASSIGNEE(S): Cancer2 Inc., USA
SOURCE: PCT Int. Appl., 42 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001003708	A1	20010118	WO 2000-US18580	20000707
W: CA				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1200104	A1	20020502	EP 2000-947094	20000707
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
US 2002148049	A1	20021017	US 2002-38035	20020102
PRIORITY APPLN. INFO.:			US 1999-142926P P	19990709
			EP 1999-113269 A	19990708
			WO 2000-US18580 W	20000707

AB Methods and compns. are provided for decreasing or regulating ratios of zinc:**cadmium** and PGE2:PGF2α and regulating the concentration of zinc-containing and PGE2-dependent matrix metalloproteinases in body fluids and tissues of a mammal, comprising administering to the mammal an amount of a pharmaceutically acceptable and bioavailable **cadmium** salt. Elevated or fluctuating levels of PGE2 and zinc and elevated concns. of zinc-containing and PGE2-dependent matrix metalloproteinases have been found to be associated with the development of certain diseases, e.g. prostate cancer, diabetes, and multiple sclerosis. Ejaculates from horse stallions showed that when the concentration of **cadmium** is increased, the sperm motility decreases. Motility of the sperm correlates to sperm viability which is an indicator of the proliferation environment of the stallion's prostate glands. Higher **cadmium** values from semen decreases the proliferation of prostate cells and replication of viruses within the prostate environment. Thus elevating the **cadmium** concentration in man's prostate gland will decrease man's incidence of prostate cancer, decreases his fertility, and protects against viral infections and age-onset diseases.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI **Cadmium** containing compositions for prevention and treatment of prostate cancer

IN **Woods, Gordon L.**

AB Methods and compns. are provided for decreasing or regulating ratios of zinc:**cadmium** and PGE2:PGF2α and regulating the concentration of zinc-containing and PGE2-dependent matrix metalloproteinases in body fluids and tissues of a mammal, comprising administering to the mammal an amount of a pharmaceutically acceptable and bioavailable **cadmium** salt. Elevated or fluctuating levels of PGE2 and zinc and elevated concns. of zinc-containing and PGE2-dependent matrix metalloproteinases have been found to be associated with the development of certain diseases, e.g. prostate cancer, diabetes, and multiple sclerosis. Ejaculates from horse stallions showed that when the concentration of **cadmium** is increased, the sperm motility decreases. Motility of the sperm correlates to sperm viability which is an indicator of the proliferation environment of the stallion's prostate glands. Higher **cadmium** values from semen decreases the proliferation of prostate cells and replication of viruses within the prostate environment. Thus elevating the **cadmium** concentration in man's prostate gland will decrease man's incidence of prostate cancer, decreases his fertility, and protects against viral infections and

age-onset diseases.

ST **cadmium** zinc salt prostaglandin prostate cancer

IT Body fluid
(**cadmium** containing compns. for prevention and treatment of prostate cancer)

IT Estrogens
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**cadmium** containing compns. for prevention and treatment of prostate cancer)

IT Estrogens
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(conjugated; **cadmium** containing compns. for prevention and treatment of prostate cancer)

IT Drug delivery systems
(inhalants; **cadmium** containing compns. for prevention and treatment of prostate cancer)

IT Prostate gland
(neoplasm, inhibitors; **cadmium** containing compns. for prevention and treatment of prostate cancer)

IT Drug delivery systems
(oral; **cadmium** containing compns. for prevention and treatment of prostate cancer)

IT Drug delivery systems
(parenterals; **cadmium** containing compns. for prevention and treatment of prostate cancer)

IT Antitumor agents
(prostate gland; **cadmium** containing compns. for prevention and treatment of prostate cancer)

IT 363-24-6, PGE2 543-90-8, **Cadmium** acetate 551-11-1, PGF2α 7440-43-9D, **Cadmium**, salts, biological studies 7440-66-6D, Zinc, salts, biological studies 10108-64-2, **Cadmium** chloride 10124-36-4, **Cadmium** sulfate 10325-94-7, **Cadmium** nitrate 37205-61-1, Protease inhibitor 149845-06-7, Invirase 155213-67-5, Ritonavir 157810-81-6, Indinavir sulfate 159989-65-8, Nelfinavir mesylate
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**cadmium** containing compns. for prevention and treatment of prostate cancer)

IT 141907-41-7, Matrix metalloproteinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**cadmium** containing compns. for prevention and treatment of prostate cancer)

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=> d his

     (FILE 'HOME' ENTERED AT 10:18:41 ON 17 SEP 2004)

     FILE 'TOXCENTER' ENTERED AT 10:20:01 ON 17 SEP 2004
L1        138461 S CADMIUM OR CD
L2         30769 S L1(L)TOXIC?
L3          3171 S L2(L)(HUMAN? OR MEN OR MAN)
L4           897 S L3(L)(ADMINIST? OR ORAL OR PARENTERAL OR INHAL? OR INJECT? OR
L5           711 S L4(L)(AMOUNT? OR DOS? OR LEVEL? OR RANGE OR CONCENTRATION? OR
L6           449 S L5 NOT PY>=1999
L7          1659 S CADMIUM(L)TOXIC?(L)(HUMAN? OR MEN OR MAN)
L8           349 S L7(L)(ADMINIST? OR ORAL OR PARENTERAL OR INHAL? OR INJECT? OR
L9           247 S L8(L)(AMOUNT? OR DOS? OR LEVEL? OR RANGE OR CONCENTRATION? OR
L10          143 S L9 NOT PY>=1999
```

L10 ANSWER 30 OF 143 TOXCENTER COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:509591 TOXCENTER
DOCUMENT NUMBER: CIS-87-00796
TITLE: Cadmium oxide
AUTHOR(S): Anonymous
SOURCE: In: EPA Chemical Profiles, United States Environmental Protection Agency, Washington D.C. 20460, USA, Dec. 1985. 4p..
DOCUMENT TYPE: Book; (MONOGRAPH)
FILE SEGMENT: CIS
LANGUAGE: English
ENTRY DATE: Entered STN: 20021200
Last Updated on STN: 20021200

AB Chemical safety information sheet. Exposure limits: OSHA PEL (TWA) = 0.2mg **cadmium**/m|3|, ceiling limit = 0.6mg **cadmium** /m|3|, TWA limit for **cadmium** oxide fume = 0.11mg/m|3|; ACGIH (1980) TLV = 0.05mg/m|3| (dust and **cadmium** oxide), ceiling limit for **cadmium** oxide fume = 0.05mg/m|3|; IDLH (NIOSH, 1978) = 0.04mg **cadmium**/m|3|. Lethal exposure for **man** has been established at 50mg **cadmium**/m|3| for 1h for **cadmium** oxide dust and for 30min for the fume. These **concentrations** may be **inhaled** without sufficient discomfort to warn workers of exposure. **Toxic** effects: tracheobronchitis, pneumonitis, pulmonary oedema, kidney and lung damage.

ACCESSION NUMBER: 1993:164941 TOXCENTER
COPYRIGHT: Copyright 2004 ACS
DOCUMENT NUMBER: CA11913132938X
TITLE: Cadmium, metallothionein and renal tubular toxicity
AUTHOR(S): Nordberg, M.; Jin, T.; Nordberg, G. F.
CORPORATE SOURCE: Dep. Environ. Hyg., Karolinska Inst., Stockholm, S-104 01, Swed..
SOURCE: IARC Scientific Publications, (1992) Vol. 118, No. Cadmium in the Human Environment, pp. 293-7.
CODEN: IARCCD. ISSN: 0300-5038.
COUNTRY: SWEDEN
DOCUMENT TYPE: Journal
FILE SEGMENT: CAPLUS
OTHER SOURCE: CAPLUS 1993:532938
LANGUAGE: English
ENTRY DATE: Entered STN: 20011116
Last Updated on STN: 20020917

AB A review with 18 refs. **Cadmium**-induced nephrotoxicity develops at **cadmium concns**. in the renal cortex of 10-300 μg/g wet weight The actual concentration at which it develops depends on a number of factors, e.g., exposure route, chemical species of **cadmium administered**, rate of **administration** and simultaneous exposure to other metals. The role of these factors can be explained by a mechanism of **cadmium** nephrotoxicity in which both extracellular and intracellular metallothionein binding play an essential role. In reindeer used for **human** food, **cadmium** was shown to be bound to metallothionein-like proteins. If **cadmium** bound to such proteins enters the blood plasma via the gastrointestinal tract, this is of special **toxicol**. significance. Metallothionein-bound **cadmium** in the plasma of exptl. animals is efficiently transported to the kidney. Tubular dysfunction in the kidney following a normally tubulotoxic **dose** of **cadmium** bound to metallothionein was prevented by preinduction of metallothionein synthesis by small nontoxic **doses** of **cadmium**

ACCESSION NUMBER: 2002:622919 TOXCENTER
DOCUMENT NUMBER: RISKLINE-1994100029
TITLE: Cadmium
AUTHOR(S): Anonymous
SOURCE: WHO Food Additives Series, (1989) 24 163-219.
FILE SEGMENT: RISKLINE
LANGUAGE: English
ENTRY DATE: Entered STN: 20021200
Last Updated on STN: 20021200

AB Comments and evaluation. Since the previous evaluation a large number of reviews have been carried out and these have been considered by the present Committee. **Cadmium** is a pollutant which affects many environmental sectors. The general population is exposed to **cadmium** principally from food and water. Although water is not a major contributor to **cadmium** intake for most individuals, elevated natural **cadmium levels** in water can occur and resultant **cadmium** intakes can be as large as the dietary contribution. Food normally represents the major source of **cadmium** exposure and available data indicate that the current intake of **cadmium** from the diet is most commonly 10-35 ug/day. Non-food sources may also be a source of **cadmium**, e.g. smoking 20 cigarettes per day may contribute a further 1-4 ug/day. **Cadmium** is a metal with an extremely long biological half-life in **man**. Even low exposure **levels** may, in time, cause considerable accumulation, especially in the kidneys. The kidney has been identified as the critical organ in relation to chronic exposure to relatively low **levels** of **cadmium** (Task Group on Metal **Toxicity**, 1976) and in particular the renal cortex. The critical tissue **concentration** of **cadmium** at which renal injury occurs is subject to inter- individual variation and the Population Critical **Concentration** (PCC) has been applied in relation to a specific response rate (e.g., PCC50 = the **concentration** at which 50% of the **human** population studied have reached their individual critical **concentration**). In relation to **cadmium**, the first adverse functional change is usually a low molecular weight (LMW) proteinuria, and intakes in the **range** of 140-255 ug/day have been associated with increased LMW proteinuria in the elderly. LMW proteinuris is not accompanied by any specific histological changes and the pathological significance of this finding is unclear. However, it can be used as an indicator of the threshold of a possible **toxic** effect and it is appropriate to set the PTWI on the basis of the **dose**-response data for this end-point. Using the concept of PCC, there are limited data on which to base an evaluation, particularly since the **concentrations** of **cadmium** in the renal cortex may fall when proteinuria occurs. Evidence of renal dysfunction in animal studies generally has been seen at renal cortex **concentrations** of 200- 400 mg/kg, but there is evidence of effects at even lower **concentrations**. In **humans** with nor, or only slight, changes with few exceptions, between 100 and 450 mg Cd/kg and studies aimed at determining critical **concentrations** in the renal cortex have yielded estimates of about 200 mg Cd/kg for the PCC10. This, of course, does not represent a no-effect-**level**. Using **dose**-response analysis for individual critical **concentrations**, a 10% prevalence rate for LMW proteinuria would be estimated to occur after 45 years exposure to dietary intakes of 200 ug Cd/day for a 70 kg person. From regression analysis of **cadmium** intake and mean kidney **cadmium concentration** in various countries, essentially similar estimates result i.e. the PCC10 of 200 mg Cd/kg renal cortex would be attained after a dietary intake of 175 ug Cd/day for 50 years. As LMW proteinuria can be demonstrated among people over 50 at a daily intake of 140-255 ug/day, this confirms that these estimates are reasonable and that there is only a relatively small safety margin between exposure in the normal diet and those which produce effects. In view of estimates that daily intake of 100 ug Cd/day would lead to about 2% of the population exceeding their individual critical **concentration**, **levels** of **cadmium** in foods and total diet should continue to be monitored and should not rise further. The Committee reiterated its previously stated position that the use of

cadmium-plated utensilsin food processing or preparation should be discouraged and galvanized equipment should be avoided where possible. Likewise, leachable **cadmium** in enamel and pottery glazes may be a source of contamination and **cadmium**-based pigments and stabilizers should not be used in food contact plastics. The use of phosphate fertilizers and sewage sludge on agricultural land may be a significant source of **cadmium** and, in some circumstances this use could lead to elevated **levels** in crops. Attempts should be made to minimize accumulation in the crops from such agricultural sources of **cadmium**. in order that **levels** of **cadmium** do not exceed 50 ug/g in renal cortex, assuming an **absorption** rate of 5% and a daily excretion of 0.005% of body burden, total intake should not exceed about 1 g/kg bw/day continuously for 50 years. The provisional tolerable weekly intake for **cadmium** was therefore set at 7 ug/kg bw. Since the PTWI is derived from estimated accumulation of **cadmium** over a period of 50 years at an exposure rate equivalent to 1 ug/kg bw/day for adults, excursions above this figure may be tolerated provided that they are not sustainable for a long period of time and do not produce a significant increase in integrated lifetime **dose**. In particular, it is recognized that this exposure will not be uniform with age. The Committee noted that the estimate of the PTWI does, in fact, take into account the higher **cadmium** intake on a body weight basis by infants and children. It is recommended that biological monitoring of groups exposed to relatively high **levels** of **cadmium** should be carried out with a view to providing supplementary data to that obtained from estimates of dietary intake.

32,33

39,45,80

88

143

AN 1994:269105 CAPLUS
DN 120:269105
TI Iron nutrition influence on cadmium accumulation by Arabidopsis thaliana (L.) Heynh.
AU Rodecap, Kent D.; Tingey, David T.; Lee, E. Henry
CS ManTech Environ. Technol., Inc., USEPA, Corvallis, OR, 97333, USA
SO Journal of Environmental Quality (1994), 23(2), 239-46
CODEN: JEVQAA; ISSN: 0047-2425
DT Journal
LA English
AB Greenhouse expts. were conducted to determine whether Arabidopsis thaliana (L.) Heynh., a putative Fe-efficient species, accumulated higher concns. of Cd from a sparingly soluble **Cd** source (**cadmium** dihydrogen phosphate) when growing in Fe-deficient rather than in Fe-sufficient conditions. The Arabidopsis plants, which were grown in double-container, vermiculite-hydroponic plot culture systems and were provided with nutrient solution containing either sufficient (89.5 μM) or deficient (0 μM) Fe supplied as the diethylene triamine pentacetate chelate, were exposed to four levels of Cd (nominally 0, 0.45, 0.89, or 1.78 mmol kg-1 vermiculite). At each substrate Cd level, rosette Cd concns. were similar at both Fe levels, but racemes and seeds from the Fe-deficient treatment accumulated significantly higher concns. of Cd than those from the Fe-sufficient treatment. For example, at the highest substrate Cd concentration, the Cd bioaccumulation factor (tissue Cd concentration predicted from polynominal response surface equations describing the relationship between tissue and substrate Cd concns. divided by nominal substrate Cd concentration) for rosettes was 3.2 in both Fe treatments, but raceme Cd bioaccumulation factors were 1.7 and 0.9, and seed Cd bioaccumulation factors were 0.4 and 0.2 in the Fe-deficient and Fe-sufficient treatments, resp. Rosette biomass was largely unaffected by tissue Cd levels, but at tissue Cd concns. of at least 1.26 mmol kg-1 for racemes and 0.32 mmol kg-1 for seeds, substantial biomass redns. occurred. Anal. of the uptake of other elements by Arabidopsis suggests that the greater accumulation of Cd by plants in the Fe-deficient treatment may be a consequence of the species' Fe efficiency mechanisms. The authors' results support the hypothesis that uptake of **toxic** elements by Fe-efficient species can be enhanced when the plants are growing in Fe-deficient soils.

AN 2002:783676 CAPLUS
DN 138:34422
TI Effect of Cadmium on Bromosulfophthalein Kinetics in the Isolated Perfused Rat Liver System
AU Soto, Armando; Foy, Brent D.; Frazier, John M.
CS ManTech Environmental Technology, Wright Patterson Air Force Base, OH, 45433-7400, USA
SO Toxicological Sciences (2002), 69(2), 460-469
CODEN: TOSCF2; ISSN: 1096-6080
PB Oxford University Press
DT Journal
LA English
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB Bromosulfophthalein (BSP) is a relatively nontoxic organic anion used as an in vivo indicator of liver performance. Elimination of BSP via the biliary system following iv injection requires dissociation from albumin in plasma, translocation across the sinusoidal membrane, conjugation with glutathione within the hepatocyte, translocation across the bile canalicular membrane, and excretion in bile. The effects of cadmium (Cd), an in vivo hepatotoxicant in rats, on BSP kinetics in the isolated perfused rat liver (IPRL) were studied to investigate the interaction between liver **toxicity** and BSP kinetics. Livers were isolated from male Fisher 344 rats. After a 30-min period for acclimation to the IPRL system, livers were dosed with **Cd** (as **cadmium** acetate), in the presence of 0.25% bovine serum albumin, to give initial concns. of 10 and 100 μM. Sixty min after Cd dosing, the IPRL system was dosed with BSP to give an initial concentration of 150 μM and the elimination kinetics of BSP from the perfusion medium were monitored. Cadmium concns. in livers at the end of the expts. were 60±4 and 680±210 μmol/kg for the 10 and 100 μM doses, resp. Exposure to 10 μM Cd for 60 min resulted in a reduction in bile flow, no significant effect on lactate dehydrogenase (LDH) leakage, and slight effects on BSP clearance. Similar studies following exposure to 100 μM Cd showed a dramatic decrease in bile flow with complete cholestasis 60 min after Cd addition LDH leakage into perfusion medium at the end of the experiment was less than 10%, indicating that Cd affected bile production well before the liver showed significant signs of necrosis. Clearance of BSP from the perfusion medium was dramatically reduced. Taken together, the data indicate that Cd has a significant effect on the kinetics of BSP in the IPRL and the dominant effects were mediated through the cholestatic effect of Cd.

AN 1998:84188 CAPLUS
DN 128:137317
TI Effects of interaction between cadmium and selenium on hepatic metabolism in mice. Part II: enzymic activity and ultrastructure
AU Skowerski, Mariusz; Czechowicz, Kazimierz; Konecki, Janusz; Jasik, Krzysztof
CS 2nd Department of Cardiology, Silesian Medical University, Katowice, 40-635, Pol.
SO Medical Science Monitor (1997), 3(5), 648-653
CODEN: MSMOFR; ISSN: 1234-1010
PB Medical Science International Publishing
DT Journal
LA English
RE.CNT 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB The histochem. methods of determining enzyme activity have provided new interesting information on the metabolism of hepatocytes in Cd, Se and Cd-Se intoxication. The activities of the following enzymes were assayed in the mouse hepatocyte: succinate dehydrogenase (SDH), Mg2+-ATPase (Mg2+-ATPase), alkaline phosphatase (AP) and acid phosphatase (AcP). Three-month long expts. were conducted on 24 male white Balb-y mice. The animals were divided into four groups. The control group I (C) was fed a standard Murigran diet. Group II (Cd) received 50 ppm **Cd** as **cadmium** acetate in drinking water. Group III (Se) was fed a diet supplemented with 5.0 mg Se/kg DM/24 h. as acid sodium selenate. Mice in group IV /Cd + Se/ received a diet supplemented with Cd and Se in the same amts. as Groups II and III. During the exptl. period mice were intoxicated with 0.2. mg Se/kg/24 h and 16.8 mg Cd/ kg/24 h. Our expts. indicated that prolonged Cd intoxication significantly lowered the activity of SDH and Mg2+-ATP-ase, which are involved in oxidative phosphorylation and active membrane transport. Accordingly, it seems that energetic processes were deranged. Selenium did not affect enzymic activities. Se interaction with Cd suppressed the **toxic** effects on the level of SDH and Mg2+-ATPase activities. Mouse hepatocytes after Cd, Se and Cd-Se-interaction treatment demonstrated ultrastructure changes which appeared to correlate with the enzymic activities noted and with the previously described incorporation of the protein synthesis precursor. The authors concluded that prolonged Cd intoxication significantly decreased the activity of enzymes involved in oxidative phosphorylation and active transport. Se in interaction with Cd suppressed **toxic** effects on SDH and Mg2+-ATPase activity.

L1 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1995:476260 CAPLUS
DN 122:238563
TI Speciation of particulate-bound cadmium of soils and its bioavailability
AU Krishnamurti, G. S. R.; Huang, P. M.; Van Rees, K. C. J.; Kozak, L. M.; Rostad, H. P. W.
CS Saskatchewan inst. Pedol., Univ. Saskatchewan, Saskatoon, Saskatoon, SK, S7N 5A8, Can.
SO Analyst (Cambridge, United Kingdom) (1995), 120(3), 659-65
CODEN: ANALAO; ISSN: 0003-2654
PB Royal Society of Chemistry
DT Journal
LA English
AB A modified sequential chemical extraction procedure was developed for partitioning particulate Cd into eight fractions: exchangeable, carbonate-bound, metal-organic complex-bound, easily reducible metal oxide-bound, organic-bound, amorphous mineral colloid-bound, crystalline Fe oxide-bound, and residual. Results of exptl. data on 16 surface soils of Saskatchewan, widely varying

in physico-chemical properties, indicate the presence of little exchangeable **Cd, cadmium** in these soils was predominantly in the form metal-organic complex-bound, accounting for 31-55%, with an average of 40%, of the total Cd present in the soils. The average relative abundance of the different forms of Cd present in these soils is in the order: metal-organic complex-bound (0.107 mg kg-1) > carbonate-bound (0.052 mg kg-1) > residual (0.042 mg kg-1) > organic-bound (0.035 mg kg-1) > crystalline Fe oxide-bound (0.016 mg kg-1) > easily reducible metal oxide-bound (0.010 mg kg-1) > amorphous mineral colloid-bound (0.002 mg kg-1). Statistical treatment of the Cd availability index, measured as ammonium hydrogen carbonate-diethylenetriaminepentaacetic acid (ABDTPA)-extractable Cd, with different particulate-bound Cd species showed high correlation (r=9.16, p = 6 + 10-7) of the Cd availability index with the metal-organic complex-bound Cd. The beta coeffs. obtained from the multiple regression anal. have given an insight into the importance of Al-organic complex-bound Cd species in estimating the bioavailability of Cd in these soils. The relationship of the metal-organic complex-bound Cd and the mobility and bioavailability of soil Cd merits in-depth research in explaining the **toxicity** and food chain contamination of Cd in the environment.

AN 1995:229947 CAPLUS
DN 122:3094
TI Effect of cadmium (CdCl2) on cell proliferation and production of EDRF (endothelium-derived relaxing factor) by cultured human umbilical arterial endothelial cells
AU Kishimoto, Takuji; Oguri, Tetsuhisa; Ohno, Mika; Matsubara, Kazuo; Yamamoto, Kazuhiko; Tada, Manabu
CS Department Environmental Medicine, Shimane Medical University, Izumo, 693, Japan
SO Archives of Toxicology (1994), 68(9), 555-9
CODEN: ARTODN; ISSN: 0340-5761
PB Springer
DT Journal
LA English
AB The effect of cadmium chloride (CdCl2) on cell proliferation and EDRF (endothelium-derived relaxing factor) production by cultured **human** umbilical arterial endothelial cells (HUAECs) was investigated. The viability of HUAECs decreased dose-dependently after the addition of **Cd** (**cadmium** chloride). Morphol. examination by phase contrast microscopy revealed severe damaging effects of Cd at higher concns. The cytotoxic effect of Cd on DNA synthesis was also concentration-dependent. The effect of Cd on EDRF production by indomethacin-treated HUAECs was assessed by its anti-platelet aggregatory effect. Platelet aggregation studies were carried out in cuvettes lined with HUAECs using an aggregometer. The anti-platelet aggregatory effect was decreased dose-dependently by pretreatment with Cd. These findings suggest that HUAECs are susceptible to concentration-dependent Cd cytotoxicity, and that Cd can inhibit the production of EDRF by HUAECs.

AN 2000:370646 CAPLUS
DN 133:146093
TI Effects of interaction between cadmium and selenium on heart metabolism in mice: the study of RNA, protein, ANP synthesis activities and ultrastructure in mouse heart
AU Skowerski, Mariusz; Jasik, Krzysztof; Konecki, Janusz
CS 2nd Clinic of Cardiology, Silesian Medical University, Katowice, Pol.
SO Medical Science Monitor (2000), 6(2), 258-265
CODEN: MSMOFR; ISSN: 1234-1010
PB Medical Science International Publishing
DT Journal
LA English
RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB Heavy metals tend to occur in increasingly many aspects of **human** activities. Studies of Cd have revealed that it is extremely toxic in its effects. It is known that Se may suppress deleterious effects of Cd. The authors investigated the effects of dietary Cd intoxication on the incorporation of precursors of RNA, protein, and ANP (atrial natriuretic peptide) granule synthesis in mouse cardiomyocytes and compared them with the results of Se interaction with Cd intoxication. Functional condition of the heart was evaluated on the basis of the number of ANP granules synthesized in cardiomyocytes of the right atrium in the mice exposed to the tested elements. The experiment was conducted on 100 male white Balby mouse during the period of 3 mo. The animals were divided into 4 groups. The control group I (C) was fed a standard Murigran diet. Group II (Cd) received 50 ppm **Cd** as **cadmium** acetate in drinking water. Group III (Se) received a standard diet supplemented with 5.0 mg Se/kg DM/24 h as acid sodium selenate. The exptl. animals in group IV (Cd + Se) were fed a diet supplemented with Cd and Se in the same amts. as the above groups. The results revealed that after a 3-mo long intoxication with Cd, 3H-uridine and 3H-alanine uptakes to cardiomyocytes were decreased by 33% and 40%, resp., and fewer ANP granules were synthesized when compared with the controls. Ultrastructure of myocytes proved slightly distorted. Se-intoxicated cardiomyocytes indicated diminished incorporation of RNA synthesis precursors by 17% and 27% in the ventricle and atrium, resp., in comparison with the controls. Some Se-induced structural changes were observed Finally, after Se interaction with Cd intoxication, the uptake of 3H-uridine and 3H-alanine to cardiomyocytes was higher and the number of ANP granules increased. The values approximated those in the controls. Thus, prolonged Cd intoxication disturbed intercellular metabolism by damaging ultrastructural elements and suppressing the incorporation of precursors of RNA, protein, and ANP granule synthesis. Se interaction with Cd revealed protective effects against Cd toxicity. After Cd-Se-intoxication, neither metabolic activity nor cardiomyocyte ultrastructure showed significant differences from those in the controls.

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L3 ANSWER 40 OF 42 MEDLINE on STN
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DOCUMENT NUMBER: 77134661 PubMed ID: 402929
TITLE: Balance study of twenty trace elements during total parenteral nutrition in man.
AUTHOR: Jacobson S; Wester P-O
SOURCE: BRITISH JOURNAL OF NUTRITION, (1977 Jan) 37 (1) 107-26. Journal code: 0372547. ISSN: 0007-1145.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197705
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AB 1. Balances of twenty trace elements (silver, arsenic, gold, bromine, **cadmium**, cobalt, chromium, caesium, copper, iron, mercury, lanthanum, molybdenum, rubidium, antimony, scandium, selenium, samarium, tungsten and zinc) have been determined in four male patients during total parenteral **nutrition** incliding fat emulsion and a special solution for addition of Fe, Zn, manganese, Cu, fluorine and iodine, besides calcium and magnesium, to the infusion solutions. 2. The analyses for trace elements were made with the aid of an ion-exchange technique based on neutron activation, and combined with subsequent gamma spectrometry. 3. The intended intravenous supply of trace elements correspond approximately to the analysed supply. However, all the other trace elements determined were found to be unintentionally administered in small amounts. 4.There was a substantial retention of Fe. Other elements retained were Ag, Co, Cr, Cu, Sb, Sc, and W. 5. Particularly Br and Rb were lost by the patients, but negative balances were also found for As, Au, Cd, Cs, Mo, Se and Zn. However, Zn was retained by one patient with short bowel syndrome. 6. The serum concentrations of thirteen (Ag, Br, Co, Cs, Cu, Fe, Hg, Mo, Rb, Sc, Se, W and Zn) of the trace elements were found to have some decrease during the period of total parenteral nutrition, mostly in accordance with the corresponding balance values, Fe, in particular, was found to have the derectional change in concentration. 7. The administration of trace elements is recommended in long-term total parenteral nutrition.

AB 1. Balances of twenty trace elements (silver, arsenic, gold, bromine, **cadmium**, cobalt, chromium, caesium, copper, iron, mercury, lanthanum, molybdenum, rubidium, antimony, scandium, selenium, samarium, tungsten and zinc) have been determined in four male patients during total parenteral **nutrition** incliding fat emulsion and a special solution for addition of Fe, Zn, manganese, Cu, fluorine and iodine, besides calcium and. . .